Clinical Pediatrics
2016, Vol. 55(3) 219–227

DOI: 10.1177/0009922815594358
cpj.sagepub.com
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Adverse Health Effects of Benzene Exposure Among Children Following a Flaring Incident at the British Petroleum Refinery in Texas City

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Abstract

This study examined the health effects of benzene exposure among children from a flaring incident at the British Petroleum (BP) refinery in Texas City, Texas. A total of 899 children (benzene exposed, n = 641 and unexposed, n = 258), aged <17 years, were included. Hematological analysis showed that white blood cell ($\times 10^3/\mu L$) counts were significantly decreased in the exposed children compared with the unexposed children (7.1 ± 2.2 versus 7.6 ± 2.1, *P* = .001). Similarly, the hemoglobin (g/dL) levels were decreased significantly in the exposed group compared with the unexposed group (12.7 ± 1.3 vs 13.1 ± 1.5, *P* = .001). Conversely, platelet ($\times 10^3/\mu L$) counts were increased significantly in the exposed group compared with the unexposed group (318.6 ± 79.8 versus 266.9 ± 58.8, *P* = .001). Hepatic enzymes were also significantly elevated among exposed children compared with the unexposed children. These findings suggest that children exposed to benzene are at a higher risk of developing both hepatic and bone marrow–related disorders.

Keywords

benzene poisoning, benzene exposure, blood disorders, chemical exposure, health impact, hematological toxicity, hepatotoxicity, refinery exposure

Introduction

Benzene is a major constituent of petroleum and occurs naturally in crude oil.[1] As a volatile organic compound, benzene is one of the main contributors to air pollutants in the environment.[2,3] Petroleum refining industries are the main sources of benzene and other toxic chemicals such as toluene, nitric oxides, and carbon monoxide. It is found in the environment as a contaminant from both natural processes and human activities.[4,5]

Human exposure to benzene has been associated with a range of adverse health effects, specifically its toxic effects on the blood and bone marrow.[6,7] It is well known that benzene exposure is associated with the risk of serious blood disorders, including leukemia, lymphoma, aplastic anemia, pancytopenia, and chromosomal aberrations.[6-10] In addition, exposure to benzene can cause a wide range of noncancerous adverse effects associated with functional aberration of vital systems such as nervous, immune, hematological, hepatic, renal, cardiovascular, and respiratory systems.[3,11-15] Thus, communities surrounding petroleum refineries have significant health risks due to the increased probability of being exposed to benzene and other toxic chemicals.

Although the health effects of benzene exposure have been very well established in adult subjects, there is paucity of information about the health consequences of benzene exposure in children. The literature on health effects of benzene in children is scant. However, several epidemiological studies have demonstrated that environmental benzene exposure is potentially a major cause of childhood leukemias.[16-18] It is well known that children are more sensitive than adults to the effects of toxic pollutants including benzene due to their differences in physiology, immaturity of enzyme systems and clearance mechanisms.[19-22] Especially, the pharmacokinetics of benzene may differ widely between children and adults due to children's incomplete metabolic systems, rapid tissue regeneration, immature host defenses, activity patterns, and high rates of infection by respiratory

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pathogens.[19,20] In addition, children have a higher unit body weight exposure than adults because of their heightened activity patterns, different ventilation tidal volumes and frequencies, which increases their exposures to toxic pollutants. Moreover, children are more susceptible to leukemogenesis because their hematopoietic cell populations are differentiating and undergoing maturation. Furthermore, the toxicodynamic processes that determine exposure rates, absorption, metabolism, excretion, and tissue vulnerability all seems to be age related[21] and thus, children are more susceptible to the effects of toxic chemicals.

In 2010, a flaring incident at the British Petroleum (BP) refinery facility led to the release of a massive amount of toxic chemicals into the air in Texas City, Texas.[23,24] Consequently, more than 500 000 pounds of toxic chemicals, including more than 17 000 pounds of benzene was released into the air threatening the health of the surrounding communities.[23-25] The Galveston County District Clerk's Office estimated that more than 50 000 people were presumed to be affected by the incident. To assess the potential health effects of ambient benzene exposure resulting from the BP flaring incident, a series of studies were conducted examining the health of the affected communities. The pilot study findings indicated that benzene exposure resulting from the BP flaring incident significantly altered the hematological and hepatic indices in the benzene exposed children compared with those of unexposed children.[26] To further substantiate these preliminary findings, we conducted a larger scale study and assessed the health effects of benzene exposure in children from this incident.

The health impact of benzene exposure in children was assessed by evaluating their hematological profile involving the measurement of their white blood cell (WBC) counts, platelet counts, hemoglobin, hematocrit, blood urea nitrogen (BUN), and creatinine. These hematological indices are routinely used to monitor and evaluate the status of health, including the detection of any changes as consequences of toxicant exposure, infection, or diseases such as cancer. In addition, serum levels of alkaline phosphatase (ALP), aspartate aminotransferase (AST), and alanine aminotransferase (ALT), which reflect the hepatic function[27] were also evaluated in children. Since benzene exposure is linked to the development of multiple hematological malignancies,[6-10] we assessed its health effects by examining the hematological and hepatic profiles in the benzene exposed children and compared them with those of unexposed children.

Materials and Methods

Subjects

This retrospective study was approved by an institutional review board. Pediatric subjects aged 17 years or less were included in this study. The details of the children's selection and the procedures employed for the clinical and laboratory evaluations were reported previously.[26] Briefly, residential areas affected by the BP refinery emission due to the flaring event were initially identified and the children exposed to the emissions were selected from the affected areas of surrounding communities of Texas City, Texas (Figure 1). Specifically, these children experienced an involuntary exposure to benzene for up to 40 days following the BP refinery flaring incident that occurred on April 6, 2010 and lasted through May 16, 2010. Children who were unexposed to benzene were drawn from primary care clinics located approximately 30 to 50 miles away from the BP refinery plant.[26] Unexposed children had visited the clinic for a routine wellness checkup and were selected randomly by the primary care physician. Demographic and clinical laboratory data were collected and included in this analysis. The study was conducted according to the ethical principles of the Declaration of Helsinki. To comply with the Health Insurance Portability and Accountability Act (HIPAA), confidentiality of information was secured by utilizing text encryption, password protection, and limited personnel involvement.

Data Analysis

Medical charts of the benzene exposed and unexposed children were reviewed and the clinical data was processed for statistical analysis. Clinical data such as WBC counts, platelet counts, hemoglobin, hematocrit, BUN, creatinine, ALP, AST, and ALT levels were assessed and compared between the benzene exposed and unexposed children.

Statistics

Data from all laboratory examinations in this study were systematically collected from the subjects' medical charts and subjected to statistical analysis. Descriptive statistics were used to assess patient demographics, which included means and standard deviations for each group. Variables included were WBC, platelets, hemoglobin, hematocrit, creatinine, BUN, ALP, AST, and ALT. Student's t test was used to assess the differences between the benzene exposed and unexposed groups. The significance level was predetermined at an alpha level of .05.

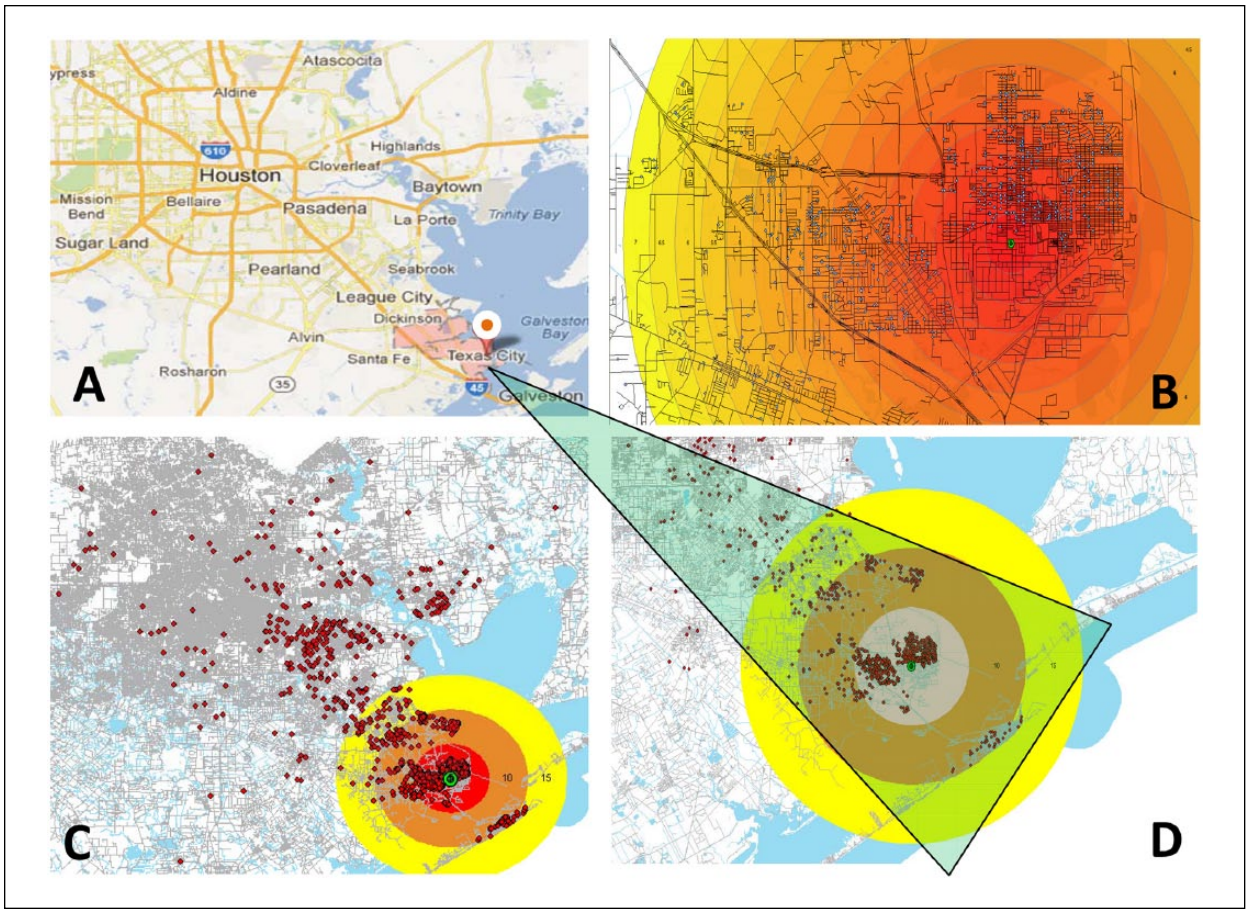

Figure 1. Map showing the location of the incident of British Petroleum (BP) refinery that spewed hundreds of thousands of pounds of toxic chemicals, including benzene and carbon monoxide, into the skies of Texas City, Texas for 40 days from April 6 to May 16, 2010. During the incident, the wind originated from south to north and dispersed the discharged benzene affecting the people residing in the northern parts of the Texas City. (A) Location of Texas City, Texas. (B) Depicted intensity of benzene exposure from BP incident surrounding neighborhoods of Texas City, Texas. The red, orange, and yellow colors depict the higher (red) to reduced (orange) to low (yellow) intensity of benzene exposure. (C) Scattered dots represent the location/address of the study participants who were exposed to benzene following a flaring incident at the BP refinery and surrounding areas. (D) A closer look at the affected area by the benzene exposure and the residential address of the study participants (scattered dots). Figure is available in full colour in the online version at cpj.sagepub.com

Results

A total of 899 children aged <17 years were included in the study. Of the 899 children, 258 were unexposed and 641 were exposed to benzene. The subjects' demographics are shown in Table 1. The mean age of the unexposed and exposed children was 10.5 and 9.5 years, respectively. Among the unexposed children (n = 258), there were 148 (57%) males and 110 (43%) females. In the benzene exposed group (n = 641), there were 334 (52%) males and 307 (48%) females. The median time from the time of the disaster to the time of laboratory testing was 156 (range, 89-526) days.

Table 1. Demographics of the Study Subjects.

Demographics	Unexposed	Benzene Exposed
Total subjects, n (%)	258 (100)	641 (100)
Mean age, years	10.5	9.5
Gender, n (%)		
Male	148 (57)	334 (52)
Female	110 (43)	307 (48)

The results presented in Table 2 show the differences in the hematologic and hepatic markers between the exposed and unexposed children to benzene. Children who were exposed to benzene experienced

Table 2. Comparison of Hematological and Hepatic Indices Between Unexposed and Exposed Children to Benzene.

Variable	Unexposed (n = 258)	Exposed (n = 641)	*P* Value
WBC ($\times 10^3/\mu L$)	7.6 ± 2.1	7.1 ± 2.2	.001[a]
Platelets ($\times 10^3/\mu L$)	266.9 ± 58.8	318.6 ± 79.8	.001[a]
Hemoglobin (g/dL)	13.1 ± 1.5	12.7 ± 1.3	.001[a]
Hematocrit (%)	38.9 ± 4.1	37.3 ± 4.0	.001[a]
BUN (mg/dL)	12.9 ± 3.5	11.9 ± 3.4	.001[a]
Creatinine (mg/dL)	0.6 ± 0.2	0.6 ± 0.2	.36[b]
ALP (IU/L)	192.1 ± 55.1	264.6 ± 64.6	.001[a]
AST (IU/L)	22.9 ± 14.6	28.2 ± 9.5	.001[a]
ALT (IU/L)	16.2 ± 6.5	20.9 ± 7.4	.001[a]

Abbreviations: WBC, white blood cells; BUN, blood urea nitrogen; ALP, alkaline phosphatase; AST, aspartate aminotransferase; ALT, alanine aminotransferase.
[a]Differences between benzene exposed and unexposed groups are significant.
[b]Did not reach statistical significance.

Table 3. Comparison of Hematologic and Hepatic Indices Between Unexposed and Exposed Male Children.

Variable	Unexposed (n = 148)	Exposed (n = 334)	*P* Value
WBC ($\times 10^3/\mu L$)	7.7 ± 2.0	7.0 ± 1.6	.001[a]
Platelets ($\times 10^3/\mu L$)	258.8 ± 56.9	313.0 ± 89.5	.001[a]
Hemoglobin (g/dL)	13.5 ± 1.8	12.9 ± 1.4	.001[a]
Hematocrit (%)	39.8 ± 4.5	37.5 ± 4.6	.001[a]
BUN (mg/dL)	13.5 ± 3.7	12.3 ± 3.3	.001[a]
Creatinine (mg/dL)	0.6 ± 0.2	0.6 ± 0.2	.40[b]
ALP (IU/L)	211.7 ± 77.6	258.2 ± 87.9	.001[a]
AST (IU/L)	23.7 ± 4.7	28.7 ± 7.0	.001[a]
ALT (IU/L)	17.1 ± 6.9	20.1 ± 9.5	.001[a]

Abbreviations: WBC, white blood cells; BUN, blood urea nitrogen; ALP, alkaline phosphatase; AST, aspartate aminotransferase; ALT, alanine aminotransferase.
[a]Differences between benzene exposed and unexposed groups are significant.
[b]Did not reach statistical significance.

significantly reduced mean WBC counts ($\times 10^3/\mu L$) compared with the unexposed children (7.1 ± 2.2 vs 7.6 ± 2.1, $P = .001$). Conversely, the mean platelet counts ($\times 10^3/\mu L$) in the benzene exposed group were significantly higher when compared with the unexposed children's group (318.6 ± 79.8 vs 266.9 ± 58.8, $P = .001$). The mean hemoglobin (g/dL) levels decreased significantly in the benzene exposed group compared with the unexposed group (12.7 ± 1.3 vs 13.1 ± 1.5, $P = .001$). Similarly, the percentage of hematocrit decreased significantly among the benzene exposed children compared with the unexposed children (37.3 ± 4.0 vs 38.9 ± 4.1, $P = .001$). BUN (mg/dL) was also found to be reduced significantly in benzene exposed group compared with the unexposed group (11.9 ± 3.4 vs 12.9 ± 3.5, $P = .001$). However, no significant differences were noted in the serum creatinine levels between the benzene exposed and unexposed groups.

Serum ALP (IU/L) levels were found to be increased in children exposed to benzene compared with the unexposed children (264.6 ± 64.6 vs 192.1 ± 55.1, $P = .001$). Similarly, the mean AST (IU/L) levels were significantly higher in the benzene exposed children compared with the unexposed children (28.2 ± 9.5 vs 22.9 ± 14.6, $P = .001$). The mean serum ALT (IU/L) levels were increased significantly in the benzene exposed group compared with the unexposed group (20.9 ± 7.4 vs 16.2 ± 6.5, $P = .001$).

The findings in Table 3 show the differences in the hematologic and hepatic markers between the benzene exposed and unexposed male children. The mean WBC counts ($\times 10^3/\mu L$) were significantly decreased in male children exposed to benzene compared with the unexposed male children (7.0 ± 1.2 vs 7.7 ± 2.0, $P = .001$). Similarly, the mean hemoglobin (g/dL), hematocrit (%), and BUN (mg/dL) levels were significantly increased in

Table 4. Comparison of Hematologic and Hepatic Indices Between Unexposed and Exposed Female Children.

Variable	Unexposed (n = 110)	Exposed (n = 307)	*P* Value
WBC ($\times 10^3/\mu L$)	8.0 ± 2.6	7.3 ± 2.1	.001[a]
Platelets ($\times 10^3/\mu L$)	277.9 ± 59.9	324.8 ± 67.4	.001[a]
Hemoglobin (g/dL)	13.1 ± 0.8	12.3 ± 1.0	.001[a]
Hematocrit (%)	37.6 ± 3.0	34.9 ± 3.2	.01[a]
BUN (mg/dL)	12.2 ± 3.0	11.6 ± 3.4	.04[a]
Creatinine (mg/dL)	0.6 ± 0.1	0.5 ± 0.1	.23[b]
ALP (IU/L)	164.5 ± 58.6	271.3 ± 74.8	.001[a]
AST (IU/L)	20.0 ± 6.0	27.6 ± 6.9	.001[a]
ALT (IU/L)	15.0 ± 5.6	20.2 ± 6.9	.001[a]

Abbreviations: WBC, white blood cells; BUN, blood urea nitrogen; ALP, alkaline phosphatase; AST, aspartate aminotransferase; ALT, alanine aminotransferase.
[a]Differences between benzene exposed and unexposed groups are significant.
[b]Did not reach statistical significance.

male children exposed to benzene compared with the unexposed male children (P = .001). Conversely, the mean platelet counts ($\times 10^3/\mu L$) were significantly increased in male children exposed to benzene compared with the unexposed male children (313.0 ± 89.5 vs 258.8 ± 56.9, P = .001). Hepatic enzymes such as ALP, AST, and ALT were also found to be significantly elevated in male children exposed to benzene compared with the unexposed male children (P = .001). However, no significant differences were noted in the serum creatinine levels between the benzene exposed and unexposed male children.

The results presented in Table 4 represent the differences in the hematologic and hepatic markers between the benzene exposed and unexposed female children. Similar to male children, female children in the benzene exposed group also experienced significantly decreased mean WBC counts ($\times 10^3/\mu L$; 7.3 ± 2.1 vs 8.0 ± 2.6, P = .001), hemoglobin (g/dL; 12.3 ± 1.0 vs 13.1 ± 0.8, P = .001), hematocrit (%; 37.6 ± 3.0 vs 34.9 ± 3.2, P = .01), and BUN (mg/dL; 11.6 ± 3.4 vs 12.2 ± 3.0, P = .001) levels compared with those in the unexposed group. The mean platelet counts ($\times 10^3/\mu L$) were significantly increased in female children exposed to benzene compared with the unexposed female children (324.8 ± 67.45 vs 277.9 ± 59.9, P = .001). Similarly, liver enzymes such as ALP, AST, and ALT in the serum were significantly increased in female children exposed to benzene compared with the unexposed female children (P = .001). No significant differences were noted in the serum creatinine levels between the benzene exposed and unexposed female children.

To evaluate if the children's age contributed to any observed health effects of benzene exposure, a further analysis was performed by grouping children into 3 (<10 years, ≥10 to <15 years, and ≥15 years) age groups and compared the clinical outcomes between benzene exposed and unexposed groups. The results in Table 5 show the differences in hematologic and hepatic markers between exposed and unexposed children among the 3 age groups. A decreased trend in the mean WBC counts was observed with increasing age in both benzene exposed and unexposed children. However, irrespective of age, the mean WBC count had significantly decreased in benzene exposed age groups compared with their matched unexposed age groups except children in age group ≥15 years. A similar trend was seen in the mean hemoglobin and hematocrit levels between benzene exposed and unexposed age groups. Conversely, benzene exposed children had significantly increased mean platelet counts compared with the unexposed children, regardless of their age group. Similarly, the serum levels of hepatic enzymes (ALP, AST, and ALT) were increased significantly in benzene exposed children compared with those of the unexposed children, irrespective of age group (Table 5).

Discussion

There is a growing body of evidence linking the exposure of benzene with multiple adverse effects including alterations in the hematologic, hepatic, immunologic, cardiac, and renal functions. Benzene exposure is also linked with pediatric cancer and intrauterine growth restriction.[19,28] Earlier studies that evaluated the potential adverse health effects of benzene have been focused mainly in adults who were exposed either occupationally or environmentally. It is well known that children are among the most susceptible populations to chemical toxicity because of their unique physical, biological, and social characteristics. In addition, multiple factors including metabolism, pharmacokinetics, and other biochemical processes, which differ substantially in children from adults may contribute to the

Table 5. Comparison of Hematologic and Hepatic Indices by Age Group Between Unexposed and Exposed Children.

Variable	Age Group (Years)	Unexposed[a]	Exposed[b]	*P* Value
WBC ($\times 10^3/\mu L$)	<10	8.1 ± 2.4	7.4 ± 2.2	.01[c]
	≥10 to <15	7.7 ± 2.0	6.8 ± 1.9	.001[c]
	≥15	6.8 ± 1.4	6.7 ± 2.2	.35[d]
Platelets ($\times 10^3/\mu L$)	<10	287.9 ± 58.5	341.3 ± 82.8	.001[c]
	≥10 to <15	263.1 ± 57.2	302.5 ± 61.7	.001[c]
	≥15	244.5 ± 53.1	278.2 ± 67.7	.001[c]
Hemoglobin (g/dL)	<10	12.7 ± 1.8	12.1 ± 1.0	.001[c]
	≥10 to <15	13.5 ± 1.0	13.0 ± 1.2	.001[c]
	≥15	13.8 ± 1.6	13.7 ± 1.5	.39[d]
Hematocrit (%)	<10	37.1 ± 3.0	35.8 ± 3.8	.001[c]
	≥10 to <15	39.6 ± 3.1	39.3 ± 3.0	.27[d]
	≥15	41.0 ± 4.7	37.8 ± 6.2	.001[c]
BUN (mg/dL)	<10	14.1 ± 3.9	11.9 ± 3.89	.001[c]
	≥10 to <15	12.3 ± 3.3	12.0 ± 3.1	.28[d]
	≥15	12.7 ± 3.2	11.2 ± 2.6	.001[c]
Creatinine (mg/dL)	<10	0.5 ± 0.1	0.5 ± 0.1	.39[d]
	≥10 to <15	0.6 ± 0.1	0.7 ± 0.1	.001[c]
	≥15	0.7 ± 0.1	0.9 ± 0.2	.001[c]
ALP (IU/L)	<10	229.2 ± 50.2	266.2 ± 75.6	.001[c]
	≥10 to <15	206.9 ± 89.4	285.7 ± 95.8	.001[c]
	≥15	198.1 ± 39.0	235.9 ± 93.7	.001[c]
AST (IU/L)	<10	26.7 ± 5.7	32.6± 8.7	.001[c]
	≥10 to <15	20.1 ± 4.8	23.4 ± 8.6	.001[c]
	≥15	17.9 ± 5.2	22.6 ± 10.4	.001[c]
ALT (IU/L)	<10	15.8 ± 5.3	20.0± 9.3	.001[c]
	≥10 to <15	15.2 ± 4.8	21.7 ± 13.7	.001[c]
	≥15	16.2 ± 7.3	23.1 ± 15.3	.001[c]

Abbreviations: WBC, white blood cells; BUN, blood urea nitrogen; ALP, alkaline phosphatase; AST, aspartate aminotransferase; ALT, alanine aminotransferase.
[a]Unexposed <10 years, n = 88; unexposed ≥10 to <15 years, n = 114; unexposed ≥15 years, n = 56.
[b]Exposed <10 years, n = 372; exposed ≥10 to <15 years, n = 168; exposed ≥15 years, n = 101.
[c]Differences between benzene exposed and unexposed groups are significant.
[d]Did not reach statistical significance.

vulnerability of children to the increased toxic effects of benzene exposure. To better understand the health effects of benzene exposure in humans, we conducted a large study involving children who were exposed to benzene following BP's flaring incident in Texas City, Texas. To the best of our knowledge, this is the first large study that has evaluated the hematological and hepatic changes found in children following benzene exposure from this incident.

The findings of this study indicate that benzene exposure induced significant changes in the hematological and hepatic profiles among children studied. In particular, the mean WBC counts were significantly decreased in children exposed to benzene compared with the unexposed children. Similarly, the hemoglobin, hematocrit, and BUN levels were decreased significantly in the benzene exposed group compared with the unexposed group. Conversely, the platelet counts were significantly increased in children exposed to benzene compared with the unexposed children. However, no significant differences were noted in the serum creatinine levels between the benzene exposed and unexposed groups.

The results of this study confirm our previous pilot study findings in which we reported that children exposed to benzene experienced significant alterations in their hematological profiles. In addition, our findings are consistent with the previously published report by Lee et al[29] on the hematological changes found in children environmentally exposed to volatile organic compounds containing low levels of benzene. The study included 192 children, of whom 97 were living near a petrochemical estate region and 95 children were living in a suburban region of Ulsan, Korea. The study showed that children exposed to low levels of benzene had significantly

decreased WBC, RBC (red blood cells), hemoglobin, and platelets than those children not exposed to benzene. However, in our study, children who were exposed to benzene had significantly elevated platelet counts compared with the unexposed children. Although the discrepancies in platelet count in benzene exposed children currently could not be explained, Ceresa et al[30] previously showed that thrombocytopenia was not a constant finding in the majority of cases resulting from chronic benzene exposure in adult subjects. Nevertheless, additional studies are warranted to clarify the effect of benzene exposure on the platelet counts in children.

Since benzene exposure has been shown to affect hepatic functions, in this study we assessed liver function enzymes such as ALP, AST, and ALT in the serum of benzene exposed children and compared them with those of unexposed children. Assessment of these liver enzyme levels in the serum is routinely used as markers for hepatic function.[31] The serum levels of ALP, AST, and ALT were found to be elevated among those children who were exposed to benzene compared with the unexposed children. These results confirm the findings of our preliminary study reported earlier.[26] Currently, there exist no studies in the literature assessing the effect of benzene exposure on liver enzymes in children to compare with our study findings. However, our results agree in part with those other published studies where increased liver enzymes have been reported in adult subjects who were exposed to benzene or petroleum products and organic solvents.[32-36] One possible explanation for increased serum levels of these enzymes could be the overproduction or release of enzymes from the liver cells in response to stimuli of hepatocellular injury or cell death. Nonetheless, our findings suggest that children exposed to benzene could be at a higher risk of hepatic tissue toxicity compared with unexposed children.

To further understand the effect of the benzene exposure by gender and age, we compared the outcomes by gender as well as by age groups (<10, 10-15, and >15 years) between the benzene exposed and unexposed children. Findings from this study indicate that both the hematological and hepatic functions were significantly affected in the benzene exposed children compared with the unexposed children regardless of the gender or age.

We acknowledge that there are several limitations to interpreting the findings of this study. Foremost, this study was conducted using cross-sectional design and therefore, the findings of this investigation should be considered carefully. It is difficult to infer a causality using such a study design because the clinical outcomes were measured at one time point after exposure to benzene. One important limitation of the study is the lack of baseline data of the children prior to the benzene exposure for comparison. Another shortcoming of this investigation is the retrospective nature of the study. Hence, further verification of the study findings is required through additional prospective randomized studies.

Regardless of the study limitations, our findings show that exposure to benzene is associated with significant adverse health effects among children. The adverse effects from benzene exposure may cause impairment not only in the hematological and hepatic functions but also in other organ functions of the children. In addition, there is significant scientific evidence that clearly links benzene exposure with an increased risk of carcinogenesis in children. It is, therefore, important that those children who were exposed to benzene be followed over time to detect further long-term toxicities to the bone marrow, liver, kidney and other organs affected by the benzene exposure as well as to monitor them for the development of secondary malignancies. In addition, organ functions including pulmonary, cardiac, neurologic, and hepatic functions should be assessed periodically to monitor the long-term adverse effects of benzene exposure in children. Periodic health checkups and routine laboratory blood work as well as pulmonary function assessment are necessary to monitor the long-term health effects of benzene exposure among children. Moreover, longitudinal and mechanistic studies on the health effects of benzene exposure are warranted to explore the importance and nature of the effects of benzene exposure in children. Since the health effects of benzene exposure appear to be long-lasting, close follow-up studies are required to determine its long-term health impact in the affected children.

Conclusion

The findings of this retrospective study indicate that benzene exposure has a potential to induce both hematological and hepatic alterations among children. Specifically, the hematological alterations included are decreased WBC counts, hemoglobin, hematocrit, BUN, and increased platelet counts in the benzene exposed children compared with unexposed children. Hepatic alterations included are increased levels of ALP, AST, and ALT in the serum of the benzene exposed children indicating hepatic injury in the benzene exposed children. It should be noted as this is a retrospective study the observed findings could be influenced by compounding factors that are inherent to the study design. Since the procedures used in this study did not follow a predefined scheme, this may have biased the interpretation of the results. Nonetheless, the findings together showed potential health effects of benzene exposure among children. Additional studies are being performed to understand the other potential health effects

of the benzene exposure from the flaring incident at the BP refinery facility in Texas City, Texas.

Author Contributions

MAD collected the data, conceptualized the study, carried out the initial analyses reviewed and edited the initial manuscript, and approved the final manuscript as submitted. GKR designed the study carried out the initial analyses, conceptualized the study, drafted, reviewed and revised the manuscript, and approved the final manuscript as submitted.

Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The author(s) received no financial support for the research, authorship, and/or publication of this article.

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